SDSU Extension

iGrow Wheat

BEST MANAGEMENT PRACTICES

Chapter 26:
Wheat Herbicide Injury

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Failure to follow a pesticide label or plants experiencing drift or tank contamination can exhibit dramatic, yet characteristic plant symptoms. If the damage occurs early and is not severe, yield loss may not occur. However, if injury occurs during a critical growth stage or is severe, the damage may result in a total crop loss. The purpose of this section is to describe and illustrate typical plant symptoms due to herbicide injury and to discuss the mechanism or mode of action of commonly used herbicides. Symptoms and images of selected herbicides are provided below.

Herbicides have been characterized by the method that the herbicide controls susceptible plants. The method is the mechanism or mode of action groups. Herbicides that have a similar mechanism of action within a plant have similar symptoms or impacts on wheat. These categories are provided in Table 26.1.

A more complete discussion is provided at http://wssa.net/Weeds/Resistance/WSSA-Mechanism-of-Action.pdf. To minimize resistance, where possible, weed management strategies should integrate herbicides with different mechanisms of action.

Table 26.1. WSSA suggested group number, mechanism-of-action (MOA), and herbicide chemistry examples.
(Chart adapted from University of Illinois, Weed Science–Extension)

MOA Group	Mechanism	Herbicide Chemistry Examples
1	ACETYL COA CARBOXYLASE (ACCase) INHIBITORS	Aryloxyphenoxypropionate (FOPs) Cyclohexanedione (DIMs) Phenylpyrazonlin (DENs)
2	ACETOLACTATE SYNTHASE (ALS) or ACETOHYDROXY ACID SYNTHASE (AHAS) INHIBITORS	Imidazolinones (Imis) Pyrimidinylthiobenzoates Sylfonylaminocarbonyltriazolinones Sulfonylureas (SU) Triazolopyrimidines
3	MICROTUBULE ASSEMBLY INHIBITOR	Benzamide Benzoic acid (DCPA) Dinitroaniline Phosphoramidate Pyridine
15	VERY-LONG-CHAIN FATTY ACID INHIBITOR	Acetamide Chloroacetamide Oxyacetamide Tetrazolinone herbicides
23		Carbetamide, chlorpropham, and propham (Note: Group 23 types of herbicides are no longer or very rarely used int U.S. crop production.)
4	SYNTHETIC AUXINS	Benzoic acids Phenoxycarboxylic acids Pyridine carboxylic acids Quinoline carboxylic acids
5	PHOTOSYSTEM II INHIBITORS Site A	Phenylcarbamates Pyridazinones Triazines Triazinones Uracils
6	Site B	Benzothiadiazinones Nitriles Phenylpyridazines
7	Site A but binds differently than Group 5	Amide Ureas
8	FATTY ACID AND LIPID BIOSYNTHESIS INHIBITORS	Phosphorodithioates Thiocarbamates
16		Benzofuranes
9	ENOLPYRUVYL SHIKIMATE-3-PHOSPHATE (EPSP) SYNTHASE INHIBITORS	Glycines (glyphosate)
10	GLUTAMINE SYNTHETASE INHIBITORS	Phosphinic acids (glufosinate and bialophos)
11	CAROTENOID BIOSYNTHESIS INHIBITORS (bleaching herbicides)	Amitrole
12		Amides Anilidex Furanones Phenoxybutan-amides Pyridiazinones Pyridines
13	Inhibits DOXP synthase	Clomazone
27	Inhibits 4-HPPD enzyme	Callistemones Isoxazoles Pyrazoles Triketones
14	PROTOPORPHYRINOGEN OXIDASE (PPG oxidase or Protox) INHIBITORS	Diphenylethers N-phenylphthalimides Oxadiazoles Oxazolidinediones Phenylpyrazoles Pyrimidindiones Thiadiazoles Triazolinones

MOA Group	Mechanism	Herbicide Chemistry Examples
17	POTENTIAL NUCLEIC ACID INHIBITORS or NON-DESCRIPT MODE OF ACTION	Organic arsenicals
25		Arylaminopropionic acids
26		Unclassified herbicides
18	DIHYDROPTEROATES SYNTHETASE INHIBITORS	Carbamate herbicide Asulam
19	AUXIN TRANSPORT INHIBITORS	Phthalamates (naptalam) Semicarbazones (diflufenzopyr)
20	CELLULOSE INHIBITORS	Nitriles
21		Benzamides
28		Triazolocarboxamides
29		Alkylazine
22	PHOTOSYSTEM INHIBITORS	Bipyridyliums
24	OXIDATIVE PHOSPHORYLATION UNCOUPLERS	Dinitrophenols (dinoterb)

4 MOA 4 – Synthetic Auxins
PHENOXYCARBOXYLIC ACIDS

Herbicide examples: 2,4-D; MCPA; Stinger®

Mechanism-of-action: Acts as a synthetic auxin, disrupting nucleic acid metabolism and protein synthesis, which ultimately leads to plant death.

Injury symptoms: Most injury occurs if applied at seedling stage or at boot stage. If applied before tillering, rolled leaves and few tillers may develop. If applied after jointing, symptoms may be twisted flag leaf, abnormal heads, and sterile spikelets (Figure 26.1). MCPA has a greater window of crop safety although high application rates or late applications may result in injury.

Wheat injury symptoms from synthetic auxins
Rolled leaves
Twisted, malformed heads
Stalk bending and brittleness
Missing kernels in spike

Injury cause
Applied to rapidly growing wheat
Applied too late

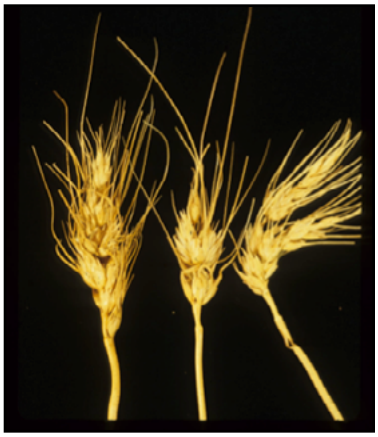

Figure 26.1. Head damage due to 2,4-D application. (Photo courtesy of Leon Wrage)

BENZOIC ACIDS

Herbicide example: Dicamba (Banvel®)

Mechanism-of-action: Acts as a synthetic auxin, see 2,4-D.

Injury symptoms: Symptoms are similar to 2,4-D. Sterile spikelets may occur if applied from the jointing to boot stage. Wheat varieties vary in sensitivity. Some exhibit no injury and some show extreme symptoms.

Images of dicamba injury: Symptoms of dicamba injury to wheat can be found at: http://www.kysmallgrains.org/productionmanual/weedmanagement.htm

Wheat injury symptoms from benzoic acid herbicides
Same as 2,4-D but may occur at lower application rates than 2,4-D

Injury cause
Variable variety sensitivity

1

MOA 1 – Acetyl CoA Carboxylase (ACCase) Inhibitors *(also known as Lipid Synthesis Inhibitors)*

Herbicide examples: Diclofop (Hoelon®); clodinafop (Discover®); fenoxyprop (Puma®)

Mechanism-of-action: Inhibits the formation of lipids used for membranes and stops growth of new tissue.

Injury symptoms: Can cause yellowing of wheat tips and blades soon after application (Figure 26.2). Browning and stunting of plants later. Applications after the jointing stage may result in stem breakage and lodging. Wet and cold conditions before or at the time of application can result in injury.

Figure 26.2. ACCase inhibitor damage.
(Photo courtesy of Leon Wrage)

9 MOA 9 – Enolpyruvyl Shikimate-3-phosphate (EPSP) Synthase Inhibitor
(also known as Amino Acid Derivative Herbicides)

Herbicide example: Glyphosate (Roundup®)

Mechanism-of-action: Amino acid synthesis inhibitor, stops synthesis of aromatic amino acids (those that contain a phenyl ring).

Injury symptoms: Yellowing on plant. Environmental conditions that slow growth (e.g., extreme heat, cold, or drought) reduce the effects of glyphosate. Youngest leaves near growing point yellow and die. If not too severe, heads may show malformation (Figure 26.3). Causes of injury may be drift from another field, misapplication after emergence, or tank contamination.

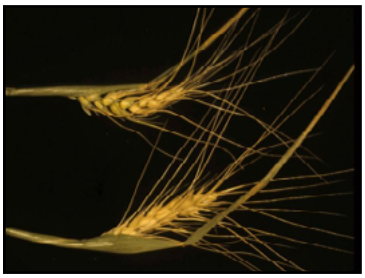

Figure 26.3. Glyphosate damage to wheat.
(Photo courtesy of Leon Wrage)

Wheat injury symptoms from EPSP synthase inhibitors
Yellow then brown foliage
Growing point dies

Injury cause
Misapplied to wheat after emergence
Tank contamination

10 MOA 10 – Glutamine Synthetase Inhibitors
(also known as Phosphoric Acid Type Herbicides)

Herbicide example: Glufosinate (Liberty®)

Mechanism-of-action: Glufosinate stops the conversion of glutamate + ammonia to the amino acid glutamine, resulting in the accumulation of toxic levels of ammonia in leaf tissue.

Injury symptoms: Symptoms appear within 3 to 5 days after treatment. Water-soaked lesions may appear and then leaves become pale yellow (such as nitrogen deficiency) or purple (may look like phosphorus deficiency).

Images of glufosinate injury: Symptoms of glufosinate injury to crops can be found in Ontario Ministry of Agricultural (OMAF) publication, "Herbicide Injury Gallery: Field Crops," online at: http://www.omafra.gov.on.ca/english/crops/facts/herbinjury_gallery/herbicidegal2.htm

Wheat injury symptoms from Glutmine synthetase inhibitors
Pale yellow or purple leaves
Water-soaked lesions

Injury cause
Misapplied or tank contamination

2 MOA 2 – Sulfonylurea (SU) Herbicides and Imidiazalinone (Imi) Herbicides

Herbicide examples: Tribenuron (Express®), thifensulfuron (Harmony®), metsulfuron (Ally XP®)

Mechanism-of-action: Both SU and Imi type chemistries of herbicides inhibit the formation of branched chain amino acids.

Injury symptoms: Injury symptoms are slow to develop with first appearance 7 to 10 days after exposure. Sensitive plants generally show overall yellowing (chlorosis) and stunting. If applied at the correct rate, injury symptoms are often temporary. For grasses, the growing point yellows and plant slowly dies. A reduction in tiller number or spike number may occur. Symptoms may be noticed even if applied according to the label rates and timings, although plants often recover with no yield loss. SU and Imi herbicides typically are applied at low rates (ounces of active ingredient per acre) and tank contamination may be a problem. In high pH soils, carryover of the SU chemistry types may be problematic, whereas carryover of Imi chemistry type herbicides is more likely in low pH soils.

Images of ALS injury: Images of symptoms to wheat can be found at: http://www.extension.umn.edu/distribution/cropsystems/components/6967_01l.html

Wheat injury symptoms from SU or IMI chemistries
Stunted plant, stunted internodes
Yellow translucent leaves
Death of growing point
Bottle brush roots

Injury cause
Variety sensitivity
Applied too late
Tank contamination

11 12 13 27 MOA 11, 12, 13, 27 – Carotenoid Biosynthesis Inhibitors *(also known as Pigment Inhibitor Herbicides)*

Herbicide examples: Isoxaflutole (Balance®), tembotrione (Laudis®), clomozone (Command®), mesotrione (Callisto®)

Mechanism-of-action: There are several herbicides with different specific mechanisms of action that are included in this group of herbicides. The common strategy among these herbicides is that enzymes in the carotenoid pigment pathway are inhibited. Carotenoids are plant pigments that include chlorophyll or protect chlorophyll from destruction.

Injury symptoms: White areas on plants or albino plants appear during emergence. Command® carryover may be seen early in the season; plants may recover from early season injury (Figure 26.4).

Wheat injury symptoms from Carotenoid biosynthesis inhibitors
White tissue
Poor emergence
Stunted plants
Growing point dies

Injury cause
Applied on cool, wet, or sandy soils
Carryover problem

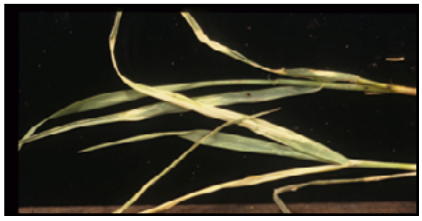

Figure 26.4. Command® injury.
(Photo courtesy of Leon Wrage)

14 MOA 14 – Protoporphyrinogen Oxidase (PPG oxidase or PROTOX) Inhibitors

Herbicide example: Carfentrazone (Aim®)

Mechanism-of-action: Inhibits protoporphyrinogen oxidase causing a cascade of events, which eventually result in cell membrane destruction and death of the plant.

Injury symptoms: Appearance of necrotic (dead tissue) speckling on leaves within a few days after exposure. Symptoms are most often observed in seedlings shortly after emergence.

Images of carfentrazone injury: Symptoms of carfentrazone injury to crops can be found at: http://weedscience.missouri.edu/herbinjsymptoms/cellmem.html

Wheat injury symptoms from PROTOX inhibitors
Yellowing or reddening of new leaves
Speckling of theolder, exposed leaves
Stunting of plant
Death of tissue and browning
Growing point dies

Injury cause
Misapplication
Tank contamination

22 MOA 22 – Photosystem I inhibitors

Herbicide example: Paraquat (Gramoxone®)

Mechanism-of-action: Herbicide accepts electrons from photosystem I and forms a herbicide radical. This radical reduces molecular oxygen to form superoxide radicals. The radicals are extremely reactive and destroy membrane fatty acids, which lead to the destruction of cell membranes and cell death.

Injury symptoms: Symptoms are often observed within within hours, especially in sunny days. Leaves develop water soaked lesions and speckling.

Images of paraquat injury: Symptoms of paraquat injury to crops can be found in University of Missouri Weed Science publication at: http://weedscience.missouri.edu/herbinjsymptoms/cellmem.html

Wheat injury symptoms from Photosystem I inhibitors
Limp leaves
Water soaked appearance (looks like frost damage)
Brown tissue in water soaked areas

Injury cause
Drift
Tank contamination

5 MOA 5 – Photosystem II Inhibitor - Triazine

Herbicide example: Atrazine (Aatrex®)

Mechanism-of-action: Stops electron flow from Q_A to Q_8 in photosystem II, which stops CO_2 fixation and production of ATP and $NADPH_2$, which are needed for plant growth. These herbicides bind at site A. Other effects include lipid and protein oxidation, which leads to leaky cell membranes and plant death.

Injury symptoms: Atrazine is not labeled on wheat, but injury may occur if there is soil carryover from the previous year (Figure 26.5 and 26.6). In addition, tank contamination from previous applications may occur. Triazine injury symptoms start as yellowing of the seedling and then death of the oldest leaves. Roots are malformed. If severe, plants will not survive.

Figure 26.5. Carryover of atrazine from previous corn crop. (Photo courtesy of Leon Wrage)

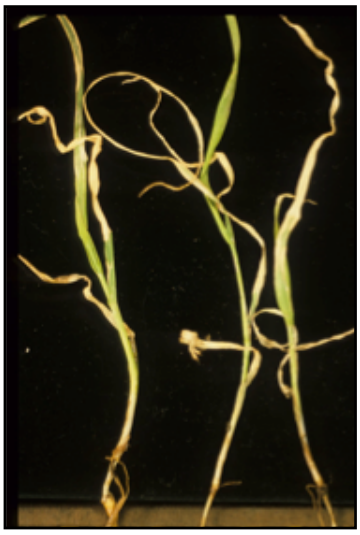

Figure 26.6. Atrazine injury. (Photo courtesy of Leon Wrage)

Wheat injury symptoms from triazine herbicides
Yellow and brown leaves

Injury cause
Cool wet conditions slowing wheat growth
Crop oil synergy if applied as a post emergence

6 MOA 6 – Photosystem II Inhibitor - Benzonitriles

Herbicide example: Bromoxynil (Buctril®, Bronate®)

Mechanism-of-action: Stops electron flow in photosynthesis in photosystem II, but unlike atrazine, binding of the herbicide occurs at site B. The final effects are similar to atrazine.

Injury symptoms: Symptoms appear as leaf tip chlorosis, general wilting, speckling and necrotic lesions to tissue where application has occurred. Young tissue that emerges after application generally is unaffected. Wheat is typically tolerant to bromoxynil (unlike atrazine), but injury may occur if cool or very high temperatures occur. Recovery is generally rapid.

Images of bromoxynil injury: Symptoms of bromoxynil injury to wheat can be found in Ontario Ministry of Agricultural (OMAF) publication, "Herbicide Injury Gallery: Field Crops," online at: http://www.omafra.gov.on.ca/english/crops/facts/herbinjury_gallery/herbicidegal2.htm

Wheat injury symptoms from benzonitrile herbicides
Yellow and brown leaves

Injury cause
Crop oil wiwth the post-emergence application

3 MOA 3 – Mitosis Inhibitor - Microtubule Assembly - Dinitroanalines

Herbicide examples: Trifluralin (Treflan®), pendimethalin (Prowl®)

Mechanism-of-action: Inhibit the growth of roots or shoots of seedlings by binding to tubulin, which leads to loss of microtubules assembly, structure, and function. This in turn leads to stoppage of cell division and cell wall formation.

Injury symptoms: Symptoms are apparent during or soon after plant emergence (Figure 26.7). Shortened, swollen root types (root clubbing), shoots are thick, short, and may be purple in color. Injury occurs if DNA herbicide is incorporated too deeply into the seeding zone. Contributing factors to increased plant injury include wet, cool soils or other stress factors such as soil compaction or drought. Carryover from a previous year's application can occur if applied late and cool conditions have occurred.

Wheat injury symptoms from dinitroanalines
Stunted plants
Roots short and thick

Injury cause
Carryover
Misapplication
Over-application

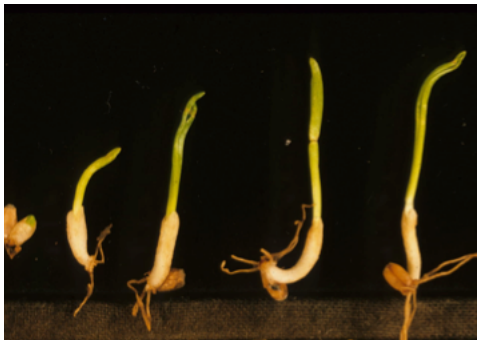

Figure 26.7. Dinitroanaline injury.
(Photo courtesy of Leon Wrage)

15 MOA 15 – Mitosis Inhibitor - Very-long chain Fatty Acid Inhibitor - Acetanilides

Herbicide examples: Metolachlor (Dual®), acetochlor (Harness®)

Mechanism-of-action: Growth inhibitor that stops the formation of very long fatty acids. This stoppage has effects on the formation of all cell membranes. Seedling roots and shoots of susceptible plants stop growing.

Injury symptoms: Death of the plant occurs soon after emergence or no plants emerge in the area.

Wheat injury symptoms from acetanilides
Poor emergence
Stunted plants
Leaf out before emergence

Injury cause
Over-application
Cool, wet soils

8 16 MOA 8 and 16 – Fatty Acid and Lipid Biosynthesis Inhibitor

Herbicide example: Triallate (Far-go®)

Mechanism-of-action: Inhibit biosynthesis of fatty acid and lipids (not through ACCase); biosynthesis of proteins; and inhibits gibberellin systhesis.

Injury symptoms: Appear during or soon after plant emergence. Reduction in cuticular wax deposition that may lead to increased disease or other stress severity. Injured seedlings may show reduced coleoptile length, stunting, or delayed emergence. Shoot tips may also fail to unroll from the coleoptiles giving the plant a buggy-whip appearance.

Images of thiocarbamate injury: Images of thiocarbamate injury on wheat can be found in the online publication, "Herbicide and Nonherbicide Injury Symptoms on Spring Wheat and Barley," University of Minnesota Extension at: http://www.extension.umn.edu/distribution/cropsystems/components/6967_01l.html

Wheat injury symptoms from triallate
Buggy whipping (leaf entrapment)
Stunted plants
Leaves emerge from the side of the coleoptile

Injury cause
Over-application
Incorporated too deeply into germinating zone of wheat
Cool, wet soils

Additional information and references

Summary of herbicide mechanism of action according to the Weed Science Society of America (WSSA). Available at http://www.wssa.net/Weeds/Resistance/WSSA-Mechanism-of-Action.pdf

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Acknowledgements

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